woman chooses to reveal such fears, worries, and concerns to the doctor.[3]

There is ample evidence of patients' frequent dissatisfaction with doctors' communication skills.[4] Despite increasingly liberal provision of information, many patients still want to know more than they are told,[5] whereas others do not want to participate in decision making: they need absolute, uncritical confidence in their doctors' skills. A skilful doctor will achieve the correct balance between autonomy and paternalism for each patient. To determine the appropriate balance it is useful to have a framework to help identify the necessary tasks and skills.

Such a framework is provided by the "three function" model of the consultation developed by Bird and Cohen-Cole[6]: gathering data to understand the patient; developing rapport and responding to the patient's emotions (to enable the patient to feel understood); and patient education and behaviour management. These functions relate to the three purposes and effects of communication: informative (to exchange information); promotive (to bring about action); and evocative (to arouse certain feelings)[7] and also to the three domains (cognitive, affective, and psychomotor) widely used by educationalists to categorise educational objectives. Each function has specific objectives and demands specific explicit skills of the doctor if they are to be achieved. Fortunately, considerable evidence now exists that such skills can be successfully acquired.[8]

However, no single model can fully convey the complexity of the doctor-patient relationship and the three function model needs to be expanded by drawing on other concepts of the consultation. These include the idea of the consultation as a "meeting between experts"[9]; the patient-centred clinical method described by Stewart et al[10]; the problem based approach[11]; the stages of motivational interviewing described by Miller and Rollnick[12]; and the family systems approach, which emphasises the importance of taking into account the patient's family and social networks.[13]

Finally, the individuality of the professional cannot be ignored. All sorts of factors, some based on the professional's own life experiences, can both consciously and unconsciously influence his or her behaviour and decisions. This has led to the notion of the doctor as a drug,[14] with both powerful effects and side effects. When listening and talking to patients, professionals need to be aware not only of the words they use, both to discover and convey information, but also their own feelings and how to cope with them.

Thus a recommendation to change terminology, while laudable in its intentions, may not be enough to alleviate mothers' dissatisfaction with the care that they receive. The risk is that mere use of the "correct" terminology, with no attention paid to the wider aspects of a consultation, could lead to professional complacency.

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Fluoroquinolone resistance

Overuse of fluoroquinolones in human and veterinary medicine can breed resistance

BMJ 1998;317:1029–30

Fluoroquinolones are highly active, broad spectrum antibiotics with many uses in both human and veterinary medicine. As with other classes of antibiotic, however, decreased susceptibility or resistance to these agents has developed. Resistance is usually chromosomally mediated so the spread of resistant bacteria contributes to the high numbers of resistant strains reported by some institutions. Although for many infections the changes in susceptibility of the bacteria have not had an immediate clinical impact, highly resistant strains have recently emerged. For some infections and bacterial species the clinical usefulness of fluoroquinolones may be limited if their use is not curtailed.

Genetic and biochemical experiments have shown two enzymes that are the targets of fluoroquinolones.[1] In Gram negative bacteria such as *Escherichia coli*, *Pseudomonas aeruginosa,* and *Neisseria gonorrhoea*, the primary target is DNA gyrase and the secondary target DNA topoisomerase IV; in Gram positive bacteria, such as *Staphylococcus aureus* and *S pneumoniae*, the primary and secondary targets are reversed.[2] DNA gyrase and topoisomerase IV are each enzymes composed of four subunits (two A and two B) encoded by *gyrA* and *gyrB*, and *parC* and *parE* respectively. Fluoroquinolone resistant isolates usually contain one or more mutations in a small section of *gyrA* or *parC*; mutation in *gyrB* and *parE* is rare.[1] In bacteria where mutations have given rise to a resistant DNA gyrase mutations then occur in the topoisomerase IV genes (and vice versa for Gram positive bacteria) to give a highly resistant bacterium. In addition there are genes that influence the uptake of the drug into the bacterial cell and efflux pumps that can be overexpressed to enhance excretion of quinolones from the cell. This enhanced efflux in turn causes increased minimum inhibitory concentrations of several drugs, including fluoroquinolones, tetracycline, chloramphenicol, and ampicillin.[3–6] It has been suggested that mutations enhancing efflux occur as a first step, allowing the bacteria to survive such that mutations can accumulate in genes encoding the target proteins. Plasmid mediated, transferable, fluoroquinolone resistance has recently been described[7]; its mechanism of resistance and epidemiology is unknown.

Gram negative bacteria such as *Enterobacteriaceae* spp and *N gonorrhoea* are usually exquisitely susceptible to fluoroquinolones, and repeated exposure to fluoroquinolones has probably allowed the bacteria to acquire and accumulate the mutations that give rise to resistance. Highly resistant *E coli* and *N gonorrhoea* have already been described.[8 9] For gut commensals such as *E coli*, frequent exposure to fluoroquinolones during treatment of other infections, such as those of the respiratory tract, may allow the accretion of mutations and emergence of highly resistant bacteria. In susceptible hosts these then give rise to infections. Although decreased susceptibility of gonococci to fluoroquinolones has become widespread, clinical resistance remains rare, occurring only in geographical hot spots such as the Far East. Clearly clonal spread of resistant bacteria is important in the spread of fluoroquinolone resistant gonococci, so efforts to reduce transfer of the bacteria are necessary.

Some bacteria such as *Salmonella typhimurium* and *Campylobacter* species are well known zoonoses. *S typhimurium* with decreased susceptibility to fluoroquinolones and *Campylobacter* resistant to fluoroquinolones have been isolated from animals and retail poultry.[10 11] For these reasons the use of fluoroquinolones in veterinary medicine has caused concern, as such strains can infect man. A fluoroquinolone is often the drug of choice for patients who have chronic enteritis, are immunocompromised, or have extraintestinal infection. There is evidence that complicated infections caused by resistant strains of *S typhimurium* are difficult to treat; *S typhi* (not a zoonose) with reduced susceptibility to fluoroquinolones (resistant to nalidixic acid) responded more slowly to fluoroquinolone treatment—and clinical outcome was poorer.[12] Therefore, it has been suggested that nalidixic acid should be used to detect decreases in susceptibility which may herald fluoroquinolone resistance. Many patients (>60%) infected with a non-typhoidal salmonella with decreased susceptibility to fluoroquinolones or a fluoroquinolone resistant *Campylobacter* have had no prior exposure to fluoroquinolone, suggesting that the bacteria must have been ingested. However, it is unclear how many patients have received a fluoroquinolone to treat their infection and whether their clinical outcome is poor. Until the full clinical relevance and risk to human health has been determined the role of fluoroquinolones in veterinary medicine remains controversial.

Another major use of fluoroquinolones is in treating respiratory tract infections, especially with the development of agents to treat pneumococcal infections. Gram positive bacteria are inherently less susceptible to fluoroquinolones than many Gram negative bacteria, so bacteria such as *Streptococcus pneumoniae* typically require only one, sometimes two, mutations in the genes encoding the target proteins to become clinically resistant. Therefore fluoroquinolone resistance is more likely to occur. For *Staphylococcus aureus* clonal spread of these bacteria in institutions has always been a problem and now fluoroquinolone resistance has been added to methicillin resistance. Good infection control will reduce the transfer and prevalence of these strains.

Fluoroquinolones have also been used as second line agents to treat multidrug resistant tuberculosis. However, mycobacteria have also been able to acquire mutations in the genes encoding the target protein.[13] Such strains are prevalent only in areas where fluoroquinolones have been widely used, and preventing the spread of *Mycobacterium tuberculosis* will reduce the numbers of resistant bacteria isolated.

As with other antibacterial agents, initial optimism about fluoroquinolones has been tempered by the development of resistant strains. The species that have become problematic are typically those that are inherently less susceptible to this class of agents. There has been misuse and abuse, so to retain the excellent activity of this class of antibiotics and reduce the development of resistant strains and their spread fluoroquinolones should be used prudently and only where there is a clinical need. For several infections a fluoroquinolone is not necessary as a first line agent.

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